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SOME EXPERIMENTAL AND CLINICAL OBSERVATIONS CONCERNING STATES OF INCREASED INTRACRANIAL TENSION.[1]

THE MÜTTER LECTURE FOR 1901.

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Gentlemen: It is my privilege to bring before the College this evening, as the Mütter lecture, some of the results of a series of experimental observations which relate chiefly to the intracranial circulation as it is influenced by local pathological processes—a subject which, from a bibliographical standpoint, is prominently associated, among others, with the names of Haller and of Astley Cooper, of Fluorens and Majendie, of Key and Retzius, of Leyden, of Althan, of Kussmaul and of François Franck, of Naunyn, Mosso, von Bergmann, of Horsley and Adamkiewicz, of Leonard Hill, and, more recently, of Kocher.

My personal introduction to the subject was due to the interest of Professor Kocher, of Bern, and the investigations as originally undertaken at his suggestion had for their primary objective point the determination whether during an increase in intracranial tension the capillaries and smaller bloodvessels of the brain were dilated from venous stasis, or, on the other hand, whether a condition of capillary anæmia was brought about by such a state.

This question, naturally one of fundamental importance for the proper explanation of the phenomena attending states of cerebral compression, curiously enough has been the occasion of much disagreement among writers. Its elucidation promised to render many of the problems relative to the symptom-complex of "Hirndruck" much more easy of interpretation.

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[1] This paper, in substance, was presented before the College of Physicians of Philadelphia, on December 3, 1901. Its experimental basis will appear in Naunyn and Mikulicz's Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie, 1902, No. 9, under the title "Physiologische und anatomische Beobachtungen über den Einfluss von Hirnkompression auf den intracraniellen Kreislauf und über einige hiermit verwandte Erscheinungen."

Not wishing to trespass too far on your indulgence, it is my intention to avoid as far as possible theoretical grounds and the discussion of the technical details of the particular experiments, but to dwell chiefly on their bearing toward operative, clinical, and diagnostic procedures. The brevity of the hour may be an apology for the omission of an historical résumé of the subject.[1]

For the sake of convenience, and in order to avoid confusion from a terminology which has been the occasion of some misunderstanding when speaking of cerebral compression, a division into two groups of the causal processes of such a state will be made. The necessity of such a differentiation, according to von Bergmann, was first emphasized by Griesinger. These groups comprise, on the one hand, processes which occasion, for the most part, a *local compression* of the brain, with a resultant unequal distribution of intracranial tension, in contradistinction to those conditions, on the other hand, which bring about a *general compression* of the central nervous system, with an equal distribution of this tension. On clinical grounds, such pathological processes as a new-growth or abscess, a blood clot, or an abnormal and circumscribed collection of fluid in one part or another of the cerebro-spinal space, illustrate the former condition; that is, subject the brain to the effects of a *local compression.* A foreign body of this sort within the enclosing walls of the skull must be accommodated at the expense of the fluids, vascular and lymphatic, which constitute a not inconsiderable proportion of an otherwise incompressible brain mass. The practical non-compressibility of the brain tissue itself since the time of Alexander Munro's investigations has been unquestioned, and the introduction, therefore, of an intracranial foreign body is only possible through the emptying of the vascular channels in its vicinity. The pressure exerted by such a foreign body, as will be seen, is not transmitted equally throughout the cerebral chamber, and in consequence the circulatory embarrassment in corresponding degree is unevenly felt.

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[1] An interesting historical and critical introduction to the subject of the intracranial circulation will be found in Mosso's work (Ueber den Kreislauf des Blutes im menschlichen Gehirn, Leipzig, 1881), and also in Berger's recent monograph (Zur Lehre von der Blutzirkulation in der Schädelhöhle des Menschen, Gustav Fischer, Jena, 1901); and in Leonard Hill's "The Cerebral Circulation," T. & A. Churchill, London, 1896. Ito in his paper (Zur Aetiologie und Therapie der Epilepsie, Deutsche Zeitschrift für Chirurgie, 1899, Bd. lii, S. 489) gives a most complete list of bibliographical references; and more recently von Bergmann (Der Hirndruck und die Operationen wegen Hirndruck, Handbuch der praktischen Chirurgie, 1900, Bd. i. S. 189) and Kocher (Hirnerschütterung, Hirndruck, u. s. w. Nothnagel's Specielle Pathologie und Therapie, 1901, Bd. ix. 3 Theil, 2 Abtheilung, S. 81) have critically reviewed the subject of the circulation in states of compression from the experimental and pathological standpoint.

In the second group of processes, namely, those associated with *general compression*, the intracranial tension may be considered to be equalized throughout the cerebral chamber, and in certain instances, indeed, over the entire central nervous system. Those traumatic cases followed by acute cerebral œdema, which Cannon and Bullard have recently ascribed to alterations in osmotic pressure—cases of acute hydrocephalus, cases of generalized meningitis, of hemorrhage into the subdural spaces from fracture of the base—processes such as these, chiefly ones in which the pressure is transmitted primarily through the fluid contents of the cerebro-spinal spaces, may be given in illustration of states associated with a general increase in intracranial tension.

Now it is not difficult to simulate experimentally the conditions produced by these two forms of compression, and I will endeavor to illustrate as briefly as possible the simple laboratory apparatus which has been employed to this end, as well as the method of direct observation of the cortical circulation during the experiments.

As early as 1811 Ravina, an Italian experimenter, in the attempt to demonstrate the pulsation of the brain within the enclosed cranium, inserted a glass window in the skull for this purpose. Donders subsequently, in 1851, employed a similar method, as did later on Kussmaul and Tenner in their attempt to disprove the so-called Monro-Kellie doctrine that the total amount of blood in the brain cannot vary with an intact skull. Although these observations are often quoted, the method used seems to have fallen into abeyance, and I am unaware that the procedure has been utilized in the study of circulatory changes during compression. It must be confessed that considerable skepticism was felt as to its practicability, but as a matter of fact it was through this method that most important corroboratory evidence of the vascular alterations during the experiments was obtained by immediate observation of the pial circulation.

An ideal window, doubtless, would be one which could be screwed into the trephine opening made for its reception; but as the simple provisional method which was first employed sufficed for all purposes, it was retained. It consists simply of a circular disk of glass with a bevelled edge made to fit into a large trephine opening, and held in place by two screws with flattened ends so bent that they hook under the edges of the opening. Under the trephine opening a small area of dura is removed sufficient in extent to expose the pial vessels overlying a convolution and at the same time an adjoining sulcus with its contained venous radicle. As a rule, also, the trephine opening in the bone has been made so as to overlap the median line, in order to expose the longitudinal sinus, to one side of which the opening in the dura has necessarily been made. (Fig. 1.) The window, though not hermetically sealed, nevertheless answers the same purpose as such a one, since an increase of tension floats the dura up against the glass in valve-like fashion.

In a great number of my experiments, drawings of the exposed area of the cortex during different stages of compression were carefully made in color, usually with the help of a low-power lens. This was necessary in order to fix in the mind's eye the minutiæ of the smaller vessels and the ground-color of the brain. Only by such observation was it possible to carry a mental picture of the topography of the fine radicles, and consequently to observe accurately the time of their appearance and disappearance from the field of vision. (See Plate, Fig. A.)

For the production of local compression the particular form of apparatus employed was similar to that used by Horsley and Spencer in their experiments, and I believe originally adopted from François Franck.

FIG. 1.

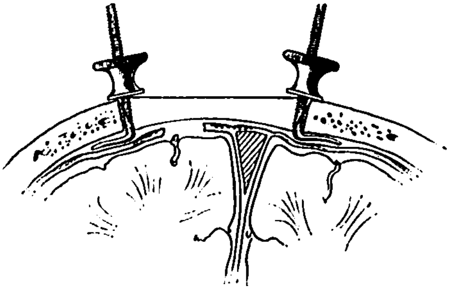
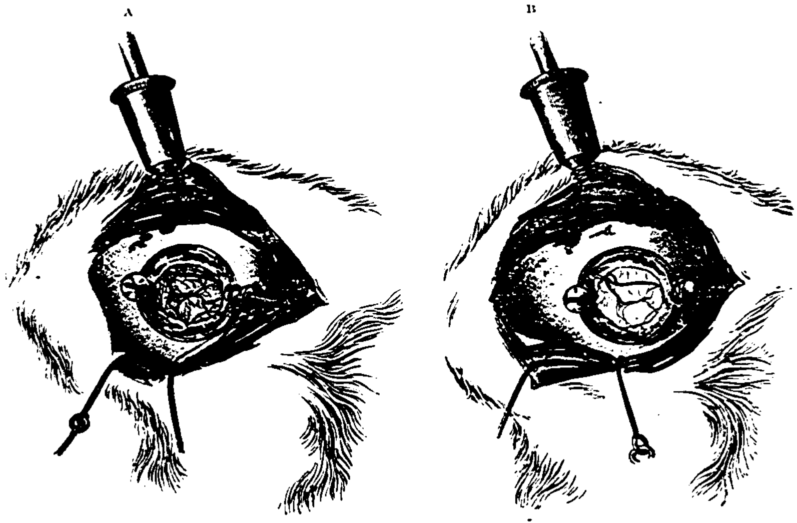


Diagram showing the cranial window in place, its relation to the longitudinal sinus, and the opening in the dura.

It consists of a double canula, the outer metal portion of which can be screwed into a trephine opening in the skull. Into this is inserted a second canula, the outer end of which communicates, by means of a heavy rubber tube of small bore, with a burette of quicksilver. To its inner or cranial end is attached a thin distensible rubber bag. (Figs. 2 and 3.)

By such an arrangement can be told not only the extent of encroachment upon the intracranial space by measurement of the amount of mercury allowed to enter the bag, but also can be told the degree of pressure necessary to evoke certain symptoms and to produce certain circulatory changes by the measurement of the height above the brain of the column of mercury in the burette. Let us suppose that this apparatus has been so introduced that pressure resultant to filling of the bag will act directly against the hemisphere without exciting sensory



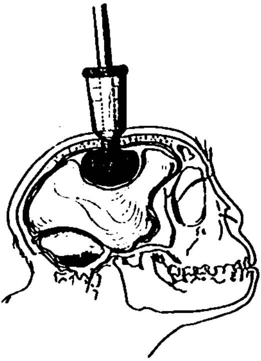
These figures, reduced one-third from original sketches, have lost much in the process of reproduction. Head of Dachshund, showing local compression apparatus and cerebral window in position. *Exp.*, *Feb.* 22, 1901.

FIG. A.—Normal appearance before increase of intracranial tension. The larger vessels should be more of an arterial color.

FIG. B.—Same animal during a period in which local compression against the right hemisphere was carried considerably in excess of the measured arterial (femoral) pressure (local pressure exerted by mercury, 110 mm.; blood-pressure equals 100 mm. of Hg.; displacement of brain produced by bag, at this time, 7 cm.; measurement of brain post-mortem 70 cm.), showing transmission of compression effects to the cortex underlying the window (left hemisphere near parietal eminence). The larger veins remain full of dark non-circulating blood and present the appearance of being broken in places. The arteries are mere threads, and should be barely distinguishable. The cortex is completely blanched.

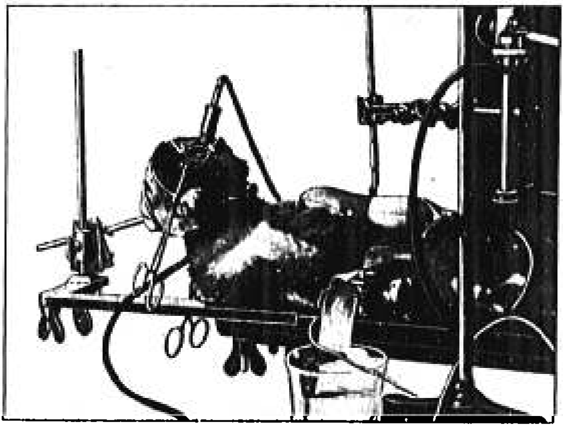
impulses from the stimulation of an interposed dura, a membrane which in the dog is especially sensitive. The first effect which will be

FIG. 2.



Semischematic representation of the method of producing local compression.

FIG. 3.



Photograph showing the cerebral window and local compression apparatus in place during an experiment.

seen to follow upon a moderate degree of compression will be a slight dislocation of the entire brain mass and a distinct widening of the smaller venous radicles exposed under the window. They at the same time become darker in color, so that their differentiation from the arteries is no longer as difficult as it is in the normal state. As the size of the foreign body increases, this venous stasis becomes apparent at remote portions of the brain, and it can be readily made out in the eye-grounds, where it seems usually to be more marked on the side of the compromising body. If the distention of the bag is further increased the intracranial tension in its vicinity finally reaches that of capillary pressure, at which time the convolutions of the brain not only in the neighborhood of the foreign body, but if the process is still further advanced even in remote parts of the hemisphere, abruptly lose their rosy color and become blanched. (See Plate, Fig. B.) The veins meanwhile remain filled with dark blood, whose means of escape seems to have been cut off, as it were, at both ends. Now, such a condition of anæmia of the hemispheres may be brought about, in the dog at all events, with but little evidence of the so-called major symptoms of compression. There usually is some slowing of the pulse and alteration in respiration, but the tolerance which an animal shows toward such an interruption of circulation in the hemispheres evidences the fact that the medulla has largely escaped from the effects of the local compression.[1]

Thus, as Horsley and Spencer, Hill, and others have emphasized, the brain does not transmit pressure equally in all directions, and so the symptoms resultant to a compression force of a local process vary greatly in accordance with the proximity of the compromising body to the vital centres of the fourth ventricle or to its remoteness from them. This rule applies to local pathological processes as well as to experimental ones in the laboratory. The counterpart, furthermore, of the laboratory experience is regularly seen on the clinical operating-table during the procedure of the elevation of a lobe of the brain such as that necessary for the removal of the Gasserian ganglion. The narrowing of the intracranial space resultant to the extensive compression and elevation of the temporal lobe at such times is productive merely of a slight slowing of pulse, with compensatory rise in blood-pressure, due to a condition of intracranial venous stasis. Such a degree of compression as is necessary to expose the ganglion, if transmitted to the medulla, would naturally be rapidly fatal. If, therefore, in clinical states some specialized centres or systems of neurones are not compromised by such a local compressing force, evidence of whose presence would thus be afforded by paralyses of one form or another, the cerebral chamber may harbor a foreign body of one-tenth to one-eighth the size of the brain without necessarily giving an indication of its presence and far from producing any of the so-called characteristic or major symptoms of compression. The venous stasis, however, which we have seen to be caused by such a condition naturally may account for some degree of apathy or stupor which usually accompanies it, and also may serve to explain the early congestion and widening of the veins of the fundus—the primary stage of the "Stauungspapille" of the Germans —in accordance with the original view of von Graefe. The symptoms produced by conditions of local compression, consequently, in so far as they are secondary to circulatory alterations, can be seen to be dependent entirely upon the position of the foreign body, what areas are involved in the local anæmia, and how far-reaching are the congestion effects.[1]

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[1] The degree to which a downward dislocation of the hind brain through the foramen magnum may account for this escape cannot be absolutely determined. That it may play some part in certain animals with conical skulls, such as rabbits and dogs, is quite possible, and attention has been called to this fact by Hill. On frozen sections of brains which have been hardened *in situ* under such circumstances it can be readily seen that the medulla may be dislocated to a considerable extent into the spinal canal. A similar dislocation I have seen to be present in slight degree in a human brain in a case in which a cerebellar cyst had finally caused fatal compression.

Owing to the irregularity, therefore, of these symptoms which this local method called out, and inasmuch as the data which we chiefly desired concerned the accurate degree of compression to which the *medulla* was subjected during the production of given symptoms, it was determined to employ some method of producing a general and equalized means of compression. In this way alone did there seem to be a possibility of accurately comparing the distinctive and peculiar symptom-complex of "Hirndruck" with the local degree of compressing force about the bulb. Such a "general compression" was produced experimentally by allowing normal salt solution, warmed to body temperature, to enter the cerebro-spinal space through a canula screwed into one part or another of the skull or in the lamina of one of the vertebræ. As a matter of fact, it was found to be immaterial, as far as the production of given symptoms was concerned, over what part of the central nervous system the fluid was primarily introduced, whether over the hemisphere, cerebellum, or cord. This fluid was allowed to enter through a rubber tube connected with a pressure flask, and would spread to all parts of the cerebro-spinal space. The tension under which it stood was regulated by the degree of elevation of the flask, and this was recorded at the same time on a revolving drum by the insertion of a mercury manometer in the course of the tube, in exactly the same way as the arterial pressure was recorded. Thus the blood-pressure and intracranial tension were written from the same abscissa, as is shown in the given chart.[1] (Fig. 4.)

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[1] According to the most recent view of v. Bergmann, if we interpret it correctly, all the symptoms of cerebral compression ("Hirndruck" in his sense) are due to the passive venous congestion which, as we have seen, is brought about by the pressure exerted by a foreign body. He believes, furthermore, that this pressure is transmitted equally over the entire central nervous system through the medium of the cerebro-spinal fluid, and consequently for equal degrees of compressing force, even in the case of a circumscribed body, that symptoms practically of equal severity would be evoked. With this view my experiments are not entirely in accord. It is possible that in some cases of local compression the escape of cerebro-spinal fluid by its natural channels may be interfered with owing to the dislocation of the brain from its normal position and the consequent obstruction of these channels of exit. Under circumstances such as these it is true that the local compression effects may be more evenly and widely distributed. In the majority of cases, however, this does not seem to be the case, and severe compression effects may be felt locally, and little, if at all, in remote territories where measurement shows that the tension of the cerebro-spinal fluid may be but little raised. Although agreeing in general with some of the principles brought forward by Adamkiewicz, a dispute in terminology over "Hirndruck" versus "Druck im Gehirn," seems rather meaningless. The writer inclines to the generally accepted view that the phenomena of compression are responsible to circulatory changes rather than to direct alterations in the nerve-tissue elements themselves from the compromising body as Adamkiewicz claims. In the states of general compression, in the sense in which we have used the term, conditions exist analogous to those claimed by v. Bergmann for all states of compression, but the process, however, must go on beyond one of venous stasis to one of vascular anæmia before the terminal symptoms of compression are produced.

Now there are certain objections which have been raised against the experimental production of an increase of tension by this method. In the first place, according to Adamkiewicz,[2] such a rapid escape of the compression fluid would take place by the normal channels of exit from the cerebro-spinal space that there would be a resultant overfilling of the right heart. Furthermore, it is objected that this escape of fluid would be incompatible with the continuance of an equalized distribution of pressure or one which is truly represented by the height of the pressure-flask.

As a matter of fact, measurement of the amount of fluid lost shows that it is not great, from 60 c.c. to 100 c.c. during a long period of compression—possibly of half an hour—which certainly is not enough to embarrass the cardiac action in the slightest degree. From many observations which I have made, however, there can be no doubt, in agreement with Adamkiewicz, but that there exists very free communication between the cerebro-spinal space and the longitudinal sinus. The original hypothesis offered by Key and Retzius, that the Pacchionian granulations subserve some purpose of filtration, can hardly be entertained; for though the points of exit of the fluid undoubtedly are in the situation of the *lacunæ laterales* of the sinus, into which these granulations for the most part project, their absence in the infant and in the lower animals would of itself be sufficient objection to this theory.[3]

Whatever the nature of these openings may be, or wherever their situation, they certainly are of considerable size and apparently possess

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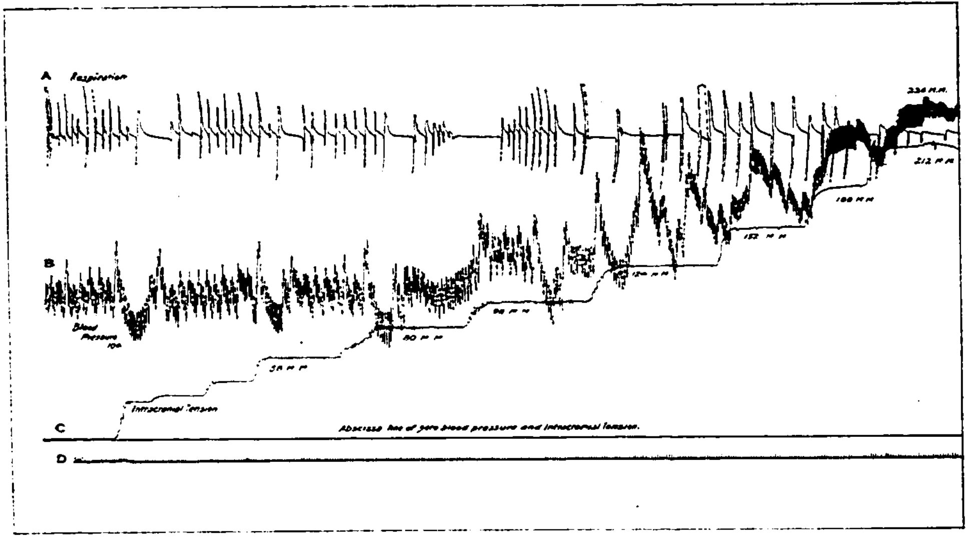
[1] A great number of these charts, whose reproduction is impossible here, were shown for purposes of demonstration. A fragment from a single one of them, taken on a slowly revolving kymograph, accompanies this article.

[2] Neurologisches Centralblatt, 1897, No. 10, vol. xvi. S. 431.

[3] The writer has had the opportunity in Prof. Sherrington's laboratory of performing hemicraniectomy and of turning down the hemisphere for the purpose of stimulating the mesial surface of the brain in the case of several anthropoid apes: gorilla, orang-outang, and chimpanzee. In no instance was there evidence of villæ from the arachnoid projecting into the dura in the neighborhood of the sinus and causing difficulty of separation of the hemisphere at this situation, as is invariably the case in adult man.

FIG. 4.



Fragment of a chart of a "general compression" experiment on a dog. Showing a gradual increase in intracranial tension from zero to 212 mm. of mercury, the blood-pressure meanwhile increasing from its normal level at 104 to 231 mm. of mercury. Note continuance of respiration, absence of vagus effects, etc.

a valvular action. The process is not a mere filtration through a membrane. This is readily shown experimentally by the fact that an injection mass entered, for example, in the lumbar meninges, may be easily made to pass from the cerebro-spinal space into the longitudinal sinus, and thence through the jugulars into the right heart. An injection in the opposite direction by way of the veins never, or but exceptionally and in small amounts, gets into the cerebro-spinal space.

The freedom of the communication of the subdural space with the sinus is demonstrated, furthermore, by the substances which will pass from one to the other. If, as occasionally happened during my experiments with local compression, the rubber pressure-bag containing mercury should rupture, and the globules of quicksilver enter the subdural space, they will be found distributed in the sinus, the jugulars, in the right heart, and throughout the lungs. I have frequently corroborated this experience on the human body by injecting mercury into the lumbar meninges, post-mortem. If the head be inverted, the skull trephined in the median line, and the longitudinal sinus opened, the mercury will be seen to pass readily from the subdural space into the sinus. To illustrate further: on many occasions the attempt was made to produce general compression by the introduction of gases under pressure instead of using the salt solution as above described. Were the gas a non-absorbable one, the animals would quickly die of cardiac air-embolism; and if the jugulars were exposed, bubbles of it could be seen pouring down toward the heart. Exposure of the cervical lymphatics and of the thoracic duct, on the other hand, showed in all instances a complete freedom from gas.

Contrary to the general belief, the longitudinal sinus, in the dog at all events, may be completely collapsed by an increase of intracranial tension. This may play an important part in the prevention of the escape of cerebro-spinal fluid under conditions such as the experimental ones of which we are speaking. It is well recognized in clinical states that the obstruction to the outflow of fluid, be it held back in the ventricles by blocking of the foramen of Majendie, or in the subdural space by inflammatory obstruction of those orifices which lead into the sinus, is especially prone to be associated with pressure phenomena. The escape of fluid under normal circumstances in the adult, whether by the olfactory, optic, or spinal nerve sheaths, plays a relatively inconspicuous rôle.

Waiving these objections, therefore, as without consequence, let us suppose, for example, that the artificial cerebro-spinal fluid be allowed to enter, by means of such an arrangement as has been described, through a trephine opening in the arch of the atlas under which the dura and arachnoid sheaths have been opened. From its proximity to the foramen magnum this would allow the fluid to spread easily up over the brain by way of the cisterna magna, and the full degree of pressure would be felt in the immediate neighborhood of the medulla. Now, in case of an increase of tension under such circumstances as this, vascular alterations similar to those which we have seen to follow upon the local compression may be observed by direct inspection of the cortex—alterations, however, much more regular in their appearance, and doubtless of corresponding degree, over the entire central nervous system. There occurs, primarily, evidence of stasis, with widening of the venous radicals and a marked color differentiation between the smaller veins and arteries. There also may be detected a distinct bluing of the rosy ground-color of the exposed convolution. Similarly, evidences of venous stasis may, in favorable cases, be made out in the fundus oculi. The eyes themselves become staring and prominent from a retrobulbar œdema, which soon spreads to the lids, and which is due to the escape of the compressing fluid along the optic sheath. This *hydrops vaginæ nervi optici* may accentuate, although I do not believe, in accordance with the theory of Manz, Schmidt, and others, that it is of itself responsible for the symptoms of the "Stauungspapille." It would seem, however, to play some part in the production of an exophthalmos. As the pressure is increased the longitudinal sinus wavers, narrows, and, from being a broad, blue band across the field of the window, thins until it is completely collapsed. Zeigler has brought forth experimental evidence to show that during an increase of intracranial tension the venous pressure in the sinus sagittalis is raised. Evidently, therefore, at some period during an early stage of compression some interference with the outflow of blood is brought about. Complete compressibility, however, of the sinus has been heretofore uniformly denied.

If I may be permitted a slight digression in order to speak for a moment of the anatomy of the venous circulation of the dog's brain, this compression of the sinus and the place where the cerebral veins themselves become obstructed may be made more clear. The large venous radicals which gather the blood from the hemisphere (the superior cerebral ones will be taken as a type of all others) come to lie in the sulci, where they are largely protected from pressure. Only in particular places do they depart from this protection in order to pass over the convex surface of a convolution. This occurs regularly at the superior longitudinal convolution, which they cross before emptying into the sinus sagittalis. At this point the veins seem to be especially subjected to the effects of compression. They enter into the sinus, for the most part, obliquely through long slits in the dura, which possess a valve-like action, so that an increase of tension in the sinus is not transmitted to the veins, and consequently an injection into the sinus, as we have stated, will ordinarily not pass back into them. The superior cerebral veins, furthermore, before entering the sinus, bifurcate, one large branch entering the diploë to join the diploëtic sinus, the other branch continuing on into the sinus sagittalis. If the sinus becomes compressed the escape of blood may take place in large amount by way of the diploë. These diplöetic sinuses in the dog are very regularly and symmetrically placed,[1] and may accommodate, presumably, the entire bulk of the venous blood from the hemispheres. Although much more developed in canine skulls, presumably sinuses of similar import exist in many cases in the calvarium of man, and under conditions of increased intracranial tension the venous pressure transmitted to them may be greatly increased. It is the experience of all operators to have encountered cases in which a craniotomy has met with an almost uncontrollable hemorrhage from the diploëtic veins. In some cases of tumor, for example, with an associated great increase in intracranial tension, the resultant increase of pressure in the veins may be so great that the bleeding from the diploë is checked with difficulty and only by plugging the openings in the bone.

Let us return once more after this digression to our experiment. During this early period of venous stasis, clinical indications of intracranial mischief doubtless would be present not only from the evidence furnished by the dilatation of vessels in the eye-grounds, but also from the mental drowsiness, apathy, or stupor which this amount of increased tension would produce. The major and characteristic symptoms of compression, however, are not evoked until a later period. As can be seen by comparison with many of these charts before you, the pressure of the fluid against the brain may be increased to the point of its equalling blood-pressure before any symptoms referable to the centres in the medulla may be called forth. Doubtless the pre-exising venous stasis may in some instances be productive of a vagus irritation, with some slowing of the pulse, indicating a nutritional disturbance of the vagal centre; but experimental evidence goes to show that the flow of blood through the vascular channels under these circumstances of stasis is no less than before, the increase of venous pressure being carried back so that the blood flows through the vessels as it might through rigid tubes. It is not until the degree of compression approximates that of blood-pressure—that is, until a condition of anæmia is approached or produced—that the major features of the pressure symptom-complex are called forth. It has usually been stated by investigators that such a degree of compression, if held, will occasion the death of the animal. The actual sequence of events does not seem to have been appreciated, for the reason that there has been no comparative measurement of the blood-pressure and the degree of intracranial tension in so far as the effects of the latter are felt in the region of the medulla.

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[1] Drawings of these sinuses are given in the Mitteilungen aus dem Grenzgebiete der Medizin und Chirurgie. Loc. cit.

The direct examination of the cortex at this period of equalization of blood-pressure and intracranial tension shows, as would be expected, an abrupt blanching of the exposed convolution. Its rosy color becomes of a grayish-yellow hue, and though the pulsating arteries themselves may be seen against this pale background, and the dark-blue veins in the sulci remain filled with blood, little if any circulation presumably passes between them. A corresponding degree of anæmia under these circumstances of "general compression" is present in the medulla, and the centres are poorly, if at all, supplied with blood.

Now, the usual consequence of this condition is not death, as commonly stated, but a stimulation of the vasomotor centre, which occasions a rise of blood-pressure sufficient to overcome the high intracranial tension. At the same time, through the cerebral window the rosy color can be seen to return again to the blanched convolution. The centres are once more nourished. Respiration, which may have ceased, is resumed, and the circulation by this regulatory mechanism has become re-established. If, after such a readjustment has taken place, the intracranial tension be once more increased and carried up to the point of again producing anæmia by exceeding the arterial tension, the blood-pressure once more rises, in its turn, to the point of exceeding the intracranial tension; so the process may be continued until the arterial pressure be forced to two or more times its normal level. The reaction is illustrated, in one way or another, in all of these charts, in some instances the pressure being held for a time as high as 250 mm. of mercury without evidence of vasomotor failure. As can be seen, on diminution of the tension the exact reverse of the process takes place, the blood-pressure taking a drop of a corresponding number of millimetres of mercury, always tending to remain, however, at a level which prevents the occurrence of anæmia at the bulbar centres.

I have endeavored to describe this reaction in its simplest form, and before speaking of the many variations which occur, let us see what is the physiological action of this response. The mechanism which calls it into play is not a difficult one to demonstrate in our animal experiments. If, in the first place, by division of both vagi we throw out of action their influence on blood-pressure and pulse-rate, and so eliminate the inhibitory action which stimulation of the vagal centre otherwise would occasion, the blood-pressure and the degree of intracranial tension, as the charts show, follow one another with even greater regularity than before. The vagus response, indeed, under other circumstances is apt to mask this simple vasomotor reaction.

If, again, the spinal cord be divided above the level of departure of the sympathetic nerves to the abdominal viscera, and thus the vasomotor control of the great splanchnic area be lost, and then cerebral compression be made, only the vagus effect will be produced, with a slowing of the pulse, but with no rise of blood-pressure whatever. If both vagi and cord are divided there is no alteration whatever in either pulse or blood-pressure on increasing intracranial tension.[1]

Similarly on cocainizing the medullary centres, and thus in another way throwing out of action both the vagus and vasomotor centres, the blood-pressure will immediately fall, and an increase of intracranial tension will not occasion the usual rise in blood-pressure—at all events, not until the cocaine effect has completely worn away. [A series of charts are here reproduced (Figs. 5-9) to show this reaction.] That

FIGS. 5-9.—Charts from a compression experiment on a rabbit (April, 1901), showing the effects of cocainization of the medulla in counteracting the usual blood-pressure response. Charts to be read from left to right. They are considerably and unequally reduced in size. *A.* Respiration which, after cocainization of medulla (Fig. 2), is continued by artificial means. *B.* Blood-pressure from femoral. *C.* Zero abscissa for both blood-pressure and intracranial tension The line is written by the pen recording the manometer level of the latter. *D.* Time.

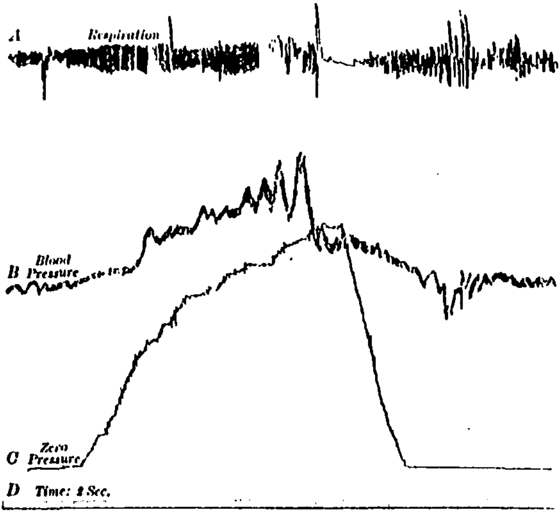

FIG. 5.—General compression experiment on a rabbit. Shows normal compression reaction. On a gradual increase of intracranial tension (*C*) to exceed the original arterial tension (*B*) the latter rises sufficiently to overcome the former. The pulse-rate is not affected, and the respiration continues. (Note that the pen recording intracranial tension (*C*) writes about 1½ cm. ahead of the blood-pressure pen (*B*).) On lowering the pressure flask to zero level the blood-pressure returns to its normal level.

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[1] Charts illustrative of these reactions have been given in the Johns Hopkins Hospital Bulletin, 1901, vol. xii, p. 290.

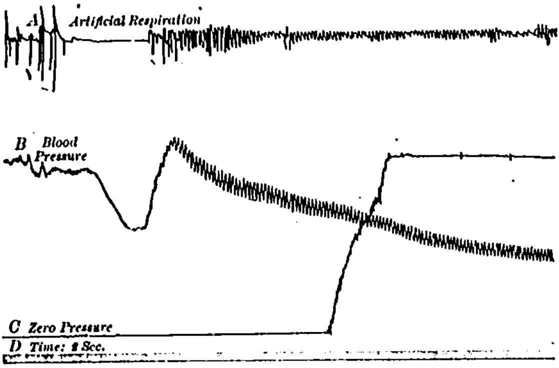

FIG. 6 shows effects upon blood-pressure in the same animal of an injection of cocaine into the neighborhood of the fourth ventricle—an immediate paralysis of respiration, with fall in blood-pressure and rapid pulse. Artificial respiration momentarily checks the blood-pressure fall. It, however, gradually drops to its paralysis level (see Fig. 7, *B*). An increase of intracranial tension (*C*) from its zero level to exceed the level of falling blood pressure is accompanied by no response on the part of the vasomotor centre. (This figure is reduced considerably more than the following ones).

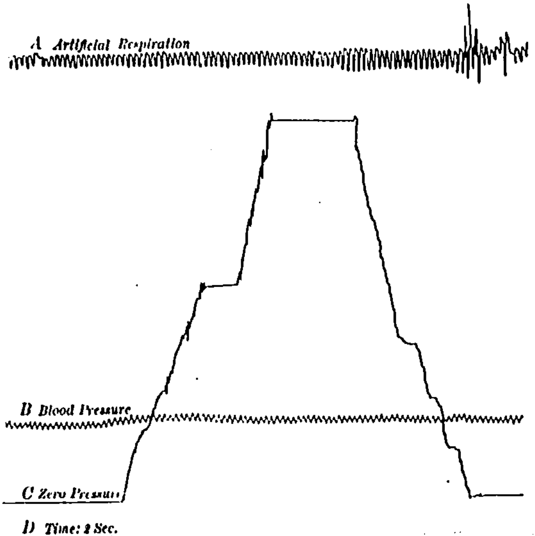

FIG. 7, showing blood-pressure (*B*) at its "paralysis level," 36 mm. of mercury, owing to vasomotor failure due to cocainization of centre. A great increase of intracranial tension (*C*) equal to 172 mm. of mercury does not affect blood-pressure to any appreciable degree.

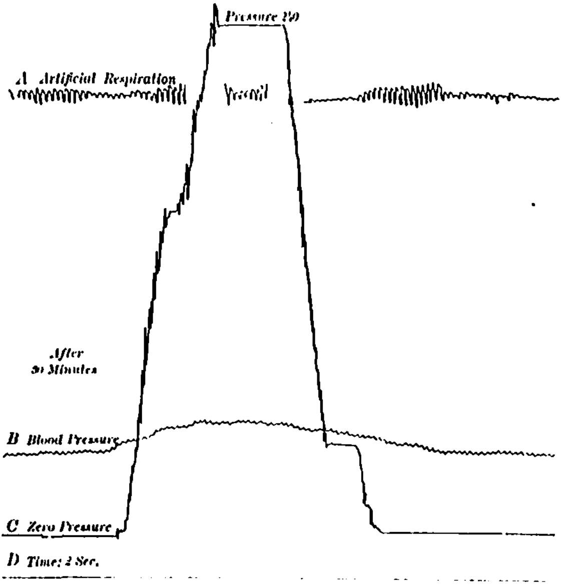

FIG. 8.—Same animal one-half hour later. Slight response on part of blood-pressure (*B*) to great increase of intracranial tension (*C*)

the mechanism is largely due to the vascular control of the great splanchnic field can be readily appreciated during an experiment by the exposure of a loop of intestine. The mesenteric and intestinal vessels can be plainly seen to diminish in calibre during the great rise in blood-pressure, and to dilate again with the fall that occurs in association with the release of the brain from the compression.

Now there are certain irregularities of interest, but of which I have avoided mention lest they confuse the description of this main underlying reaction. If, for example, the intracranial tension is *suddenly* brought to the point of equalling or of exceeding the blood-pressure, there ensue evidences of a pronounced vagus stimulation, usually with a complete checking or "Stillstand" of the heart, lasting at times over a period of thirty seconds and often associated with the production of spasms of the Kussmaul-Tenner type. If the pressure is held, however, there is finally a gradual release from this extreme vagus inhibition, the heart-beat slowly returns perhaps to its normal rate, and the blood-

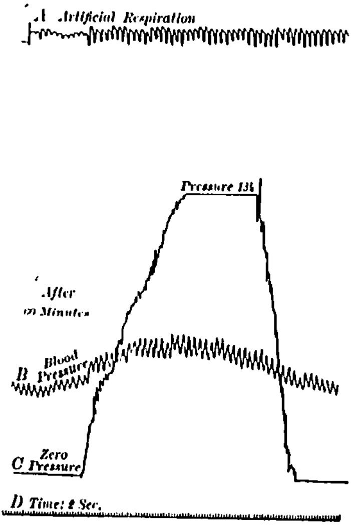

FIG. 9.—One hour later. Cocaine effects wearing away. Blood-pressure response to increase of tension more apparent.

pressure gradually ascends to or above its necessary level. Under conditions which presumably correspond more closely to the majority of those occurring in clinical states, in which there is a very much more gradual increase of tension, such a pronounced vagal irritation may be avoided, and the rise of blood-pressure will occur in anticipation of that degree of compression which would otherwise equal or exceed it, and so bring about a condition of anæmia of the bulb. Thus in many of the observations represented by these charts, by slowly increasing the intracranial tension, even though it ultimately be carried far above that corresponding to the original level of the blood-pressure, we have been able to avoid (as shown by the curves and as corroborated by the view through the cerebral window) any period of circulatory interruption in the medulla. (See Fig. 4.) Only exceptionally, however, has it been possible to obtain this vasomotor response in a fresh animal without sufficient circulatory disturbance in the neighborhood of the bulb to produce some slowing of the pulse and respiration. Usually, when the compression is occasioned with comparative rapidity (from eight to ten minutes, for example) symptoms are produced that are the experimental counterpart of the deep, stertorous respiration, slow vagus pulse, and high blood-pressure which we see clinically in corresponding cases.

Observations which we have been enabled to make on an occasional case of fracture of the base in which hemorrhage has occurred into the subdural spaces have given us exact clinical corroboration of the experimental reactions. If in these cases it has been impossible to relieve the intracranial tension by the removal of a wide area of bone, a *circulus vitiosus* of symptoms is established in the following way. There is, in the first place, an increase of intracranial tension from the effused blood. When this becomes sufficient in amount to embarrass the circulatory conditions at the medulla the familiar clinical picture is displayed, with the slowing of pulse and respiration, and the high, bounding, incompressible, peripheral artery, whose tension can be measured, at times, almost at 300 mm. of mercury. This rise in blood-pressure, which under all circumstances is a conservative one, for the furtherance of a proper nutritional supply to the vital centres, here suffices merely to increase the intracranial bleeding, and thus in a still greater degree to increase the tension which has primarily evoked the response. Under such circumstances the vasomotor centre rapidly fails.

Another characteristic, which many of these blood-pressure curves show when the vasomotor centre is straining to overcome the condition tending to produce bulbar anæmia, is that of a rhythmic or wave-like variation in pressure—the well-known Traube-Hering waves—the causation of which has been the occasion of much dispute. In these instances it can be seen that the crests of the waves rise above and their hollows fall below the level of the intracranial tension. Examination of the circulatory condition of the brain through the window at the time of their production shows that an interval of cerebral anæmia corresponds to the low-pressure stage of the wave, whereas a condition of active capillary circulation rides on their crests. These circumstances are well shown on many of these charts taken during stages of general compression experiments. When the compression exceeds the blood-pressure anæmia ensues; the vasomotor centre is stimulated; the blood-pressure, rises in these cases higher than is necessary to readjust the circulation; the bulb is well supplied with blood—perhaps too well—and is hyperæmic; the stimulated centre relaxes to too great a degree; the arterial pressure falls below the level at which blood can enter the compressed vessels; the medulla once more becomes anæmic. Thus the centre is again stimulated, and so the process is repeated indefinitely. These rhythmic waves, furthermore, in many of these charts can be seen to follow the general level of blood-pressure as this is raised or lowered by alteration in the degree of intracranial tension.

It is quite possible that these rhythmic activities are nothing more than an exaggeration of the normal rhythmic waves of blood-pressure which are known to take place under a variety of conditions. Furthermore, as has been stated, the primary object of this elevation of blood-pressure seems to be for the continuance of nourishment to that most important of all centres, the respiratory one. A period of anæmia such as occurs during the low-pressure stages of the Traube-Hering waves leads promptly, as a rule, to the cessation of respiration. As long, however, as the vasomotor centre keeps the blood-pressure higher than that of intracranial tension, and at a point sufficient to keep the medulla supplied with arterial blood, respiration continues. Thus accompanying the crests of the waves of blood-pressure can be seen respiratory efforts, which assume more or less the character of the Cheyne-Stokes type of respiration. The period of shallow respiration or of actual apnœa accompanies that stage of the wave which is associated with cerebral anæmia. Such a periodicity of respiration, as we shall see, can be demonstrated clinically in association with waves of blood-pressure; shallow or passive respiratory efforts accompanying the low stage, deep and snoring respirations the rhythmic periods of its elevation.

The length of time experimentally during which these reactions can endure is considerable, even under the extreme degree of elevation—over 200 mm. of mercury from a level of 90 or 100 mm., in many cases—to which we have pushed the blood-pressure. These extraordinary responses under clinical conditions doubtless are called out only in rapidly fatal cases, such as those of basal hemorrhage with fracture, as has already been instanced. The more slowly the intracranial space is compromised probably the longer will the response hold. In the experimental as well as in the clinical cases, unless relief is given, a period comes at which the vasomotor centre weakens, loses its grasp on the great splanchnic field, the blood-pressure falls below that of the intracranial tension, the respiratory centre is no longer nourished, and the individual dies with a low blood-pressure and rapid heart, whose action may persist for hours, provided that the respiration is continued by artificial means.

A striking instance of this kind came under my observation a short time ago—one which illustrates just such a terminal stage and also the danger of any further increase of tension during the critical period of cerebral compression with bulbar involvement. The patient entered the hospital in a condition of profound stupor, with a subnormal temperature, a snoring respiration, a heart beat of fifty to the minute, a high-tension pulse (unfortunately it was before the days of a practical blood-pressure apparatus, and I cannot give the exact figures), and a "Stauungspapille"—all indicating such an increase of intracranial tension that the bulb was in the condition bordering on the so-called

"Lähmungsstadium" of the Germans. With evidence of an old otitis media as a localizing indication, an operation was immediately performed. The abscess was difficult to find, inasmuch as it had no apparent communication with the diseased temporal bone, and in the course of our search, during the elevation of the temporal lobe which was suspected to harbor it, the respiration ceased. The medullary centre, evidently suffering already from compression effects, could not endure this additional burden. On immediately releasing the pressure, and after some artificial respiratory efforts, spontaneous breathing returned. On resuming the search the abscess was finally located and evacuated, but the respiration during the procedure had again ceased. This time, in spite of artificial respiration, spontaneous breathing was never resumed, though every possible means of reawakening it by sensory stimulation and otherwise was resorted to. The characteristic rapid pulse, of low tension, indicating a vasomotor failure, had appeared, and at this low level of blood-pressure the heart continued to beat for *twenty-three hours*, during which time artificial respiration was continued. Though a careful gross and microscopical examination was made of the medulla of this case by Dr. Paton, no lesions worthy of note could be detected. Similar instances of respiratory failure anticipating the cardiac failure by many hours have been reported by Victor Horsley, Macewen, and Hudson.

It has been heretofore pointed out that in death from a fatal increase of intracranial tension the arrest of respiration precedes that of the heart. Back of the respiratory failure, however, in the majority of cases, and responsible for it, if we may judge from experimental evidence, stands the collapse of the vasomotor regulatory mechanism, which up to this time has held the blood-pressure at a height sufficient to overcome the abnormal extravascular tension at the medulla. Prompt surgical relief, with a wide opening of the calvarium, may save life even in desperate cases with pronounced medullary involvement. Thus an extensive craniectomy may completely, though temporarily, ward off the serious attacks which accompany the increasing tension of an intracranial growth. In acute cases, on the other hand, as in those of hemorrhage accompanying basal fracture, I believe that in the majority of instances the pressure symptoms may be completely and permanently warded off.

Two years ago there was brought to the hospital a small boy who had fallen, as a child falls, head downward, from a third-story window to a basement below. His skull had been fissured in all directions, like a fallen egg-shell, and when first seen he was nearly in the "Lähmungsstadium" of compression—comatose, with a slow pulse of very high tension, a Cheyne-Stokes respiration, and convulsive spasms of the Kussmaul-Tenner type, resembling those seen during the period of cerebral anæmia in the experimental conditions. An immediate operation—laying back the tense scalp by anterior and posterior flaps, such as are made on the autopsy-table, with elevation of large plates of depressed bone and the removal of many fragments—resulted in the immediate relief of all bulbar symptoms. The brain itself, however, was so hemorrhagic and disorganized that there seemed to be little hope of its ever returning to a functional condition. For three weeks the patient lived the life of a spinal animal, unconscious and with absolutely no spontaneous movement, but with normal pulse and respiration and normal visceral reflexes, so that he would swallow when liquid was put in the back of his mouth. Not until twenty-one days of this absolutely passive state had elapsed was there evidence of the return of some cerebral activity by a slight motion in one hand and the occasional feeble elevation of the eyelids. He ultimately made a complete recovery, but had narrowly escaped at the time of operation the period of vasomotor paralysis.

It must be remembered, however, that the sudden removal of pressure from the brain when the blood-pressure has been forced to considerable heights may be followed by a paralysis instead of a release from the major compression symptoms. The occasion of this is readily brought out by post-mortem examinations, which, under such circumstances, oftentimes discloses a brain and medulla of a uniform cherry-red color, from the widespread extravasation of blood due to the multiple rupture of the minute bloodvessels. The external supporting pressure of the high intracranial tension has been suddenly removed, leaving the internal or intravascular pressure too great for the strength of the vessel walls.

An opportunity was recently offered, in Dr. Osler's wards, to follow through the paralytic stage of compression a patient for the relief of whose condition operative interference had been refused. A brief relation of the clinical observations made in this case may more closely bring into line the bedside observations with some of these experimental experiences.

The patient was brought to the hospital with the history of indefinite mental symptoms which had extended over a period of some weeks. He had become somnolent and apathetic, and had gradually lost the use of the entire left side. Examination showed an individual in stupor who would not reply to interrogation, but could be roused sufficiently so that he would show his tongue and open his eyes. The eyeballs themselves seemed somewhat prominent and the lids somewhat congested. A bilateral choked disk of advanced degree was present. There was apparently complete motor paralysis of the left arm and leg, with some anæsthesia. The right leg was also paralyzed, and the increased muscular tonicity of the right arm showed that paralysis might be expected there. There was no acceleration of the pulse (80). The respiratory rhythm was unaltered. There was no abnormal increase in blood-pressure. It was evident that some local process was present in the right brain, the tension from which was transmitted in sufficient degree to the opposite hemisphere to cause profound alterations in circulation, as shown by the pressure paralysis and by the change in the fundus oculi. No symptoms, however, had yet given evidence of any circulatory disturbance in the remote situation of the bulb. At the end of five days, with a gradually deepening stupor, the medulla began to show signs of involvement, chiefly as an increased tension of the pulse. As in the experimental conditions, when the intracranial tension about the bulb is slowly and gradually brought to the neighborhood of the blood-pressure the latter may rise to counteract its effects, without the production of the slow vagus pulse and deliberate respiration almost invariably seen in more rapid processes.[1]

At the time of this beginning period of bulbar involvement a needle was inserted in the lumbar meninges and the pressure there registered—as I believe the pressure should always be recorded in case of lumbar puncture—by connecting a long glass rod of small bore with the needle, and by measuring in this way the height to which the column of fluid rises. It should normally equal the venous blood-pressure, about 4 mm. of Hg. In this particular case the fluid rose to a height corresponding to 44 mm. of mercury. Evidently the local increase in tension was transmitting its effects, although in diminished degree, to the remotest part of the cerebro-spinal space. The blood-pressure meanwhile measured between 180 and 200 mm. of mercury. By means of the blood-pressure apparatus, furthermore, the rhythmic waves of arterial pressure could be easily made out. If the encircling band of the apparatus about the upper arm was held at a sufficient degree of tension to collapse the brachial artery, and so to obliterate the radial pulse at the wrist, after a few moments the pulse would appear for a short time and then once more fade away, but only to again reappear in this wave-like fashion. A similar and associated periodicity of respiration, furthermore, was seen to be taking place. Coincident with the crests of the blood-pressure waves occurred active periods of respiration, with at the same time a wide dilatation of the pupils and the swinging out of the eyeballs in conjugate deviation toward the side of the lesion. Coincident with the periods of low blood-pressure occurred the shallow respiratory action, and at such times there was usually an asymmetrical condition both as regards the size of the pupils and the position of the axes of the globes.

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[1] For the proper estimation of gradual alterations in arterial pressure in clinical cases reliance should not be placed alone on the palpating finger any more than that the hand should be depended upon for the determination of a patient's temperature. The arterial pressure should be spoken of in figures, just as is the temperature and pulse-rate, and not in the loose way generally employed, in which we describe it as "weak," or "thready," or "bounding," etc., as the case may be. For this purpose we should have a plotted record of the pressure, since it is of far greater importance in all conditions, surgical and medical, than is the pulse-rate which we so studiously record. There are to-day simple forms of apparatus which may be easily set up and employed for practical purposes. The particular form which has been introduced into the wards of the Johns Hopkins Hospital was demonstrated. It requires no particular familiarization for its satisfactory employment. It represents the Riva Rocci apparatus in its simplest form, and consists of a distensible rubber armlet made to encircle the arm or leg, and which may be inflated until the peripheral pulse is shut off. The degree of tension necessary to accomplish this is registered on a simple mercury manometer inserted between the armlet and the pump used for the inflation. It is true that this apparatus gives us simply a measurement of systolic pressure, and as Brush and Howell have recently emphasized, such a systolic pressure does not represent the mean blood-pressure. As a matter of fact, however, only in exceptional instances is there an inequality in the variations of systolic and diastolic pressures so marked as to be of clinical import, and the simplicity of this apparatus as a means of obtaining one of them, the systolic, renders it of inestimable value as a clinical "instrument of precision." The Mosso apparatus registering diastolic pressure, the Gärtner tonometer, and the Hill apparatus with spring manometer, all possess for clinical purposes insurmountable disadvantages.

Other and detailed analyses of symptoms I will pass over, merely wishing to draw an analogy with the experimentally induced processes of like nature. After about twelve hours of the high-tension pulse the vasomotor centre began to show signs of failure, the blood-pressure gradually dropped, the tension of the cerebro-spinal fluid in the cord fell considerably, as was told by a second lumbar puncture,[1] and the patient finally died from a respiratory failure, leaving the rapid-running, uncontrollable pulse of complete vasomotor relaxation to end the story.

Clinical corroboration of these experimental data which have been given might be drawn from a multitude of cases. An instance of the effect of local compression in the near neighborhood of the medulla was given by a recent case of cyst of the cerebellum which was seen in Dr. Osler's clinic. As has been stated, the local compression effects are most pronounced in the immediate vicinity of the compromising body. A growth in the cerebellum consequently is one which is apt to suddenly throw out of action by circulatory embarrassment the regulatory centres in the medulla. This may occur before other absolute evidence of its presence has been given, and in this particular case the diagnosis of an intracranial growth had not been definitely made. From the remoteness of the process to the ophthalmic veins the vascular disturbances of compression due to venous stasis were not transmitted to the eye-grounds, which were normal in appearance. The patient complained of some occipital headaches, of weakness, and there was some exaggeration in the deep reflexes. A lumbar puncture had been performed, evidencing but slight, if any, increase in the tension of the fluid in the spinal meninges. The patient unexpectedly one night passed into the stage of medullary involvement, with loss of consciousness, a slow vagus pulse, stertorous respiration, and a very high blood-pressure, which, unfortunately, owing to the urgency of the case, was not registered, but which was said to have occasioned an almost incompressible pulse. In a short time the vasomotor mechanism gave way, respiration ceased in conjunction with the fall in blood-pressure, and, as in the majority of

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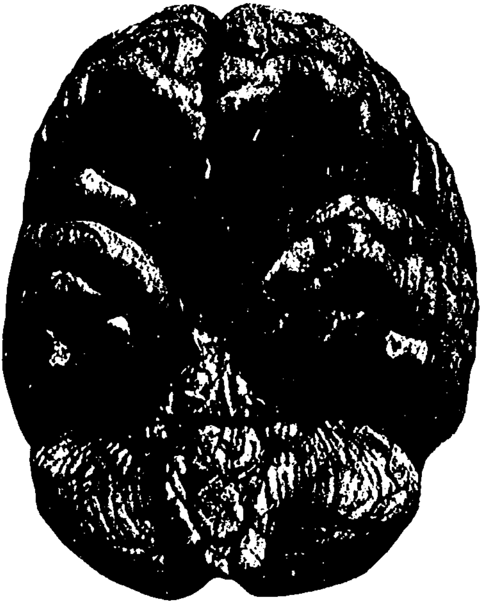
[1] Just how much stress may be laid upon the measured tension of the fluid in the spinal meninges for purposes of diagnosis and for the estimation of degrees of concomitant intracranial tension, I am unable at present to state. Certainly in localized intracranial processes it does not represent, as v. Bergmann's theory would indicate, the degree of "Hirndruck," as this case clearly evidences for a general compressing force of 44 mm. of mercury is by no means a fatal one. Presumably the dislocation downward of the brain, and consequent blocking of the foramen magnum, plays a certain part in shutting off the spinal fluid from the effects of tension existent within the cranial chambers. Nevertheless, in the cases of cerebral tumor in which I have performed lumbar puncture, there has been some abnormal increase in the tension of the spinal fluid. In the clinical states representing conditions of general compression, such as meningitis, acute cerebral œdema, subdural hemorrhage, etc., the tension of the fluid in the lumbar meninges more nearly corresponds with the intracranial tension which is producing the compression symptoms.

FIG. 10.



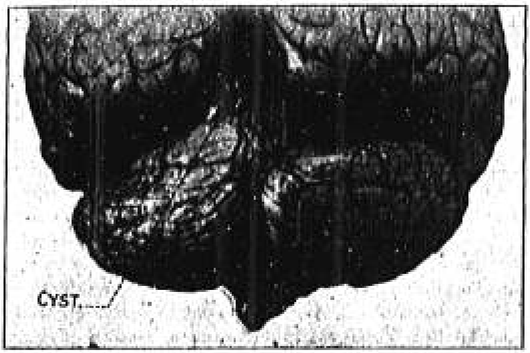
Photograph of the base of the brain of a case which died from compression effects of a cerebellar cyst. The dotted line corresponds to the imprint of the edge of the foramen magnum. A mould of the upper part of the spinal canal is made by the included portion of cerebellum and medulla.

these conditions, the heart continued to beat for some time after death had actually ensued.

The accompanying photographs (Figs. 10 and 11) show in this case the marked dislocation downward into the foramen magnum of the medulla, together with a surrounding ring of cerebellum. Whether this dislocation, as has been suggested in the case of lower animals, could allow the medulla in any way to escape from the compression effects, or whether it would actually increase their action, must remain a question of uncertainty.

There are other points of interest concerning which my curtailed time forbids making more than passing mention. The pulsation of the brain, for instance, which, on Mosso's principle of the plethysmograph, increases *pari passu* with the increase of intracranial tension, becoming most marked when the pressure from without and the blood-pressure correspond, may give the surgeon at times valuable data. When transmitted to the artificial cerebro-spinal fluid, as in the experiments, it may even exceed in the length of the manometric excursions those of the arterial pulse itself. It is perhaps worthy of note that the character and height of this pulse-wave do not correspond exactly to that of the arterial pulse.

FIG. 11.



Posterior view of same, showing cyst and portion of cerebellar mould of spinal canal.

Of interest also is the question of vasomotor nerve supply to the cerebral vessels themselves. Undoubted experimental evidence of their presence has baffled physiological investigation, although the anatomist seems to have already prepared the ground by a histological demonstration of their presence. On the clinical side also the existence of such nerves would be favored by instances such as the recurring appearance of paresis or aphasia in cases of Raynaud's disease in association with evidences of local vasomotor spasm of other territories of the body. If, however, one may draw conclusions from the mechanism of the vasomotor centre above described, a mechanism which constricts the vascular fields of the body, thus raising the blood-pressure in order to supply an anæmic medulla with blood, a corresponding narrowing of the cerebral vascular field from the same stimulus would seemingly counteract the conservative object of this mechanism. Contrary to the positive statements of many, substances like epinephrin will blanch the pial vessels over the area of its application, as will occasionally a jet of cold water against the brain or the faradic current used for cortical stimulation. Whatever part, however, the cerebral vasomotor system may play, if such a system there be, it certainly is controlled from centres and by influences other than those which call into play the vasoconstriction in the peripheral and intra-abdominal fields.

From this rather superficial review of the alterations in the cerebral circulation as they are influenced by varying degrees of compression some effects stand out with especial prominence.

In the first place, the venous stasis, which becomes apparent on but a moderate increase of the tension, fortunately gives early evidence of itself in the eye-grounds except in those cases of local compression, in posterior basic meninges, for example, so remotely situated that the compression effects are not readily transmitted as far as the cavernous sinus and ophthalmic veins. Furthermore, local pathological processes, such as are confined to the hemispheres, may be responsible for local circulatory disturbances sufficient to cause a cessation of function of a large part of the forebrain without leading in any way to a corresponding implication of the medulla. When, however, the local process is in the near proximity of, or, if remote, when its effects are so far reaching that the vital centres of the bulb are compromised, the one symptom which with regularity is called forth, and which betokens a serious alteration in the local circulation, is a persisting rise in blood-pressure, which may or may not be associated with a pronounced vagus pulse, with rhythmic alterations in blood-pressure and with a retardation or periodicities of the respiration approaching a Cheyne-Stokes type.

The first and minor symptoms of compression are found in association with varying degrees of intracranial venous stasis, the major symptoms of "Hirndruck," with an approaching capillary anæmia of the medulla.